Glomerulonephritis - Acute

**Definition**

Glomerulonephritis is an inflammatory process affecting primarily the glomerulus, with infiltration and proliferation of acute inflammatory cells. These are principally mononuclear cells and neutrophils in post-infectious glomerulonephritis. The inflammation is immunologically mediated with immune deposits in the glomerulus. Onset of symptoms is usually acute. Symptoms include oliguria, hypertension, haematuria, proteinuria and renal impairment.

Acute post-streptococcal glomerulonephritis (APSGN) is the most common glomerulonephritis affecting children. There are specific nephritogenic strains of group A streptococcus which cause acute nephritis. It is usually sporadic and is uncommon in very young children. Other infections - mycoplasma, staphylococcus, pneumococcus and viral infections (parvovirus) may also cause acute glomerulonephritis.

**Clinical Features**

- Pharyngitis precedes APSGN by 8-14 days. The time between purulent skin disease and acute nephritis is very variable.
- Severity varies from asymptomatic microhaematuria to anuric renal failure.
- Gross haematuria occurs in 30-50%.
- Volume overload occurs in up to 2/3 of patients and may be severe enough to cause congestive heart failure and pulmonary oedema.
- Hypertension occurs in up to 80%. Severity may not correlate with degree of volume overload.
- Hypertensive encephalopathy (seizures, confusion, coma) is the presenting feature in 5%.

**Laboratory Features**

- Throat swabs are rarely positive.
- Leucocytes and red cell casts in urine.
- Proteinuria is usually no more than 2+ on dipstix, however some children develop nephrotic-range proteinuria
- Mild normochromic, normocytic anaemia due to haemodilution.
- Depressed C3 complement in 90%, normalises 2-6 weeks after onset.
- Prior penicillin therapy may attenuate the fall in C3
- Urea is raised disproportionately to creatinine
- Urine sodium excretion is low compared with acute tubular necrosis
- Acidaemia and hyperkalaemia may occur in those with severely depressed renal function
Differential Diagnosis

IgA nephropathy, Alport's disease, mesangiocapillary glomerulonephritis, Lupus nephritis, Thin basement membrane nephropathy.

Management

Supportive care is all that is required in most. Bed rest does not influence rate of recovery. Antibiotics do not change the course of illness once established but should be given to reduce infectivity.

Complications

1. Hypertension

May cause seizures. Hypertension in the early stages of illness is usually due to volume overload.

We suggest treating with salt and water restriction (400ml/m2/day)

PLUS

Furosemide 2- 4 mg/kg/dose IV.

The dose needs to be titrated according to effect. Discuss with a senior if giving an IV dose > 40 mg.

If additional acute hypertensive management is needed then add:

Isradipine 0.1 mg/kg/dose as required up to q6- 8h.

This is a Hospital only medication. Will need to be changed to a different medication if hypertension persists at discharge.

Patients with severe hypertension and encephalopathy should be admitted to PICU and managed in consultation with the paediatric nephrologist and intensivist on call. (See Management of Hypertensive Emergency - Hypertension guideline).

The choice of intravenous antihypertensive drug is at the discretion of the treating physician:

IV Labetalol: Bolus of 0.2 - 1 mg/kg, followed by a constant infusion (1 mg / ml in 0.9% NaCl).

Begin infusion at 1 mg / kg / hour. Increase the infusion rate at 10 -15 minute intervals until there is an effect. If there is no effect at a dose of 2.5 mg / kg / hour, choose another agent.

IV Hydralazine: Bolus 0.1 - 0.2mg/kg (max 20mg)

If BP falls too rapidly, give boluses of normal saline.

2. Electrolyte abnormalities.

Usually not severe, potassium restriction may be required if renal function significantly reduced.
3. Volume overload

Treat with fluid restriction -400ml/m2/day. Monitor weight and serum sodium daily during acute phase.

**Indications for Renal Biopsy**

- Persistently low C3 beyond 8 weeks
- Persistent heavy proteinuria after 6 months
- Atypical presentation – nephrotic syndrome, severe acute renal failure with estimated GFR <30ml/min/1.73m²
- Atypical course – failure of renal function to improve after initial improvement during the acute phase which usually last no more than 2 weeks.

**Time course to resolution of APSGN**

![Time course to resolution of APSGN diagram]

**Clinical and laboratory features**

- Gross H
- Hypertension
- Low C3
- Persistent proteinuria
- Intermittent proteinuria
- Microhaematuria

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**Time course**